EUROPEAN JOURNAL OF BIOMEDICAL AND PHARMACEUTICAL SCIENCES
http://www.ejbps.com

ISSN 2349-8870
Volume: 8
Issue: 9
621-623
Year: 2021

METABOLIC OXIDANTS PRECIPITATE THE COMPLICATION OF DM

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Article Received on 23/07/2021 **Article Revised on 13/08/2021** **Article Accepted on 03/09/2021**

ABSTRACT

Introduction: Diabetes is a metabolic disorder of carbohydrates, protein and lipids resulting into hyperglycemia due to deficiency and absence of insulin. This disorder leads to increased production of free radicals causing causing complications of diabetes. Malon Dialdehyde (MDA) is a biomarker for free radicals mediated lipid damage and oxidative stress.

Aims and Objectives: To study the role of MDA as a primary Biomarker of diabetic complication.

Material and Methods: Study comprised of 50 each of IDDM and NIDDM patients and healthy individual controls. Samples were collected for estimation of Blood glucose and MDA.

Results:-MDA level was found to be elevated in case of Diabetes and more in case of NIDDM.

KEYWORDS: DM, NIDDM, MDA level, Blood glucose.

INTRODUCTION

Diabetes Mellitus is a common metabolic disorder of primarily carbohydrates and also protein and lipids resulting into hyperglycemia due to deficiency, absence or insensitivity of insulin with clinical presentations of polyurea, polydyspsia, polyphasia and unexplained weight loss (WHO). Biochemical parameter for the diagnosis of DM is as follows according to WHO:

Fasting Plasma Glucose- 126 or more mg/dl (normal 70-110 mg/dl)

Post Prandial or Random Plasma Glucose - 200 or more mg/dl (normal 70-140 mg/dl)

During diabetes, persistent hyperglycemia causes increased production of free radicals especially Reactive oxygen species (ROS), for all tissues from glucose auto-oxidation and protein glycosylation. Free radicals are generated as byproducts of normal cellular metabolism, however, several conditions are known to disturb the balance between ROS production and cellular defence mechanism. This imbalance can result in cell dysfunction and destruction in tissue injury.[1] Free radicals may play as important role in causation and complication of DM. In DM, alterations in the endogenous free radical scavenging defence mechanism may lead to ineffective scavenging of ROS resulting in oxidative damage and tissue injury. Oxidative stress is currently suggested as a mechanism underlying diabetes and diabetic complications.[2] Enhanced oxydative stress and changes in anti oxidase capacity, observed in both clinical and experimental DM, are thought to be etiology of chronic diabetic complications.[3] It has been reported that oxidative stress may constitute the key and common event in the pathogenesis of secondary diabetic complications.[4] Free radicals are continuously present in the body as a result of normal metabolic processes and interaction with environmental stimuli.[5] There is convincing experimental and clinical evidence that the generation of ROS increases in both types of DM (Type 1 & 2) and that the onset of diabetes is closely associated with oxidative stress (32-33). Oxidative stress results from increased ROS and/or Reactive nitrogen species (RNS) (34). Examples of ROS include charged species eg. superoxide and hydroxyl radicals and uncharged radical species eg. H2O2 and singlet oxygen. The possible sources of oxidative stress in DM might include auto-oxidation of glucose, shift in redox balance decreased tissue concentration of low molecular weight antioxidants eg. reduced glutathiom (GSH) and vitamin E and impaired activity of antioxidant defence enzyme eg. Superoxide dismutase (SOD) and Catalase (CAT).[6] Overtime, convincing evidence has established the role of free radicals and oxidative stress in the pathogenesis and development of complications in DM including Retinopathy, Nephropathy, Neuropathy and acclerated Coronary Artery Disease.[7,8,9,10]

Lipids are reported as one of the primary largest of ROS.

Hydroperoxide have toxic effects on cell both directly and through degradation to highly toxic OH- radicals. They may also react with transition metals like iron or copper to form stable aldehyde eg. Malondialdehyde (MDA) that damage cell membrane. Peroxidation of lipids produces highly reactive aldehydes, Including MDA Acrolein, 4 Hydroxy Nonenal (HNE), 4 Oxo Nonenal (ONE) and Isolevuglandins (IsoLGs).

MDA has been documented as primary biomarker of free radical mediated lipid damage and oxidative stress.

AIMS AND OBJECTIVES

This work has been designed to study of oxidative stress (MDA) in two types of DM and controls to establish the role of MDA as a primary biomarker of complication of DM.

MATERIALS AND METHODS

The study was a cross sectional study selected randomly in the department of Clinical pathology, PMCH, Patna. The subjects were chosen from the OPD in the department of medicine, PMCH, Patna. The study group comprised of fifty-fifty each IDDM and NIDDM patients and control of fifty healthy individual persons. After following the exclusion and inclusion critria, a short history and clinical examination of patients were obtained regarding the diabetes, BP, duration of DM, history of drug intake and after taking verbal consent of the patient, sample collected.

Samples were collected for estimation of

1. Blood glucose (Fasting & PP)
2. MDA

Study tool- Fully automated analyser

1. Glucose estimation – GOD- POD method

Assay procedure

Reagent 1	Blank	std	Test
	1 ml	1 ml	1 ml
standard	-	10 microL	-
Sample	-	-	10 microL
Distilled water	10 microL	-	-

Mixed, incubated for 10 minute at 37 degree C and absorbance against 500-540 nm in auto-analyser. Results were calculated by the analyser and printed out.

2. Estimation of Malondialdehyde (MDA)

MDA in the catabolite of lipid peroxide can react with thiobarbituric acid (TBA) and produce red compound, which has a maximum absorption peak at 531 nm.

Reagent composition

Reagent I - Clarificant

Reagent II – Acid Reagent

Reagent III – Chromogenic agent

Reagent IV – Standard

Operative steps

	Blank Tube	Standerd Tube	Sample Tube	Control Tube
Reagent (ml.)		a^x		
Absolute ethanol (ml.)	a^x			
Sample (ml.)			a^x	a^x
Reagent 1 (ml.)	a^x	a^x	a^x	a^x
Reagent 2 application solution (ml)	30	3.0	3.0	3.0
Reagent 3 (ml)	1.0	1.0	1.0	
50% glacial Acetic Acid (ml)				1.0

a^x represents the volume of sample, standard, absolute ethanol & reagent 1. They are equal (O.1 ml.) MDA (n mol/ml.) $\frac{OD\ sample - OD\ Blank}{OD\ Std - OD\ Blank}$ × concentration of standard (10 n.mol/ml) Control. Normal Volume 1.076 n mol/ml

RESULTS

Table 1: Mean and SD of age among case and control group.

	Mean	SD	P Value
Case (IDDM)	33.06	± 8.52	<0.001
Case (NIDDM)	46.80	±9.76	<0.001
Control	41.34	±8.34	<0.001

The mean age and SD value of IDDM Patients was 33.06 & ±8.52. In NIDDM patient, mean age & SD value was 46.8 & ± 9.76. The same for control 41.34 & ±8.34.

Table 2: Mean and SD value of MDA among case and control group.

	IDDM	IDDM	NIDDM	N IDDM	CONTROL	CONTROL	P VALUE
	Mean	SD	Mean	SD	Mean	SD	
MDA	0.76	0.26	2.52	0.39	0.49	0.16	<0.001

The above table shows mean & SD value of MDA level in IDDM, NIDDM patients and control group was 0.76 & ± 0.26, 2.52 & ±0.39, 0.49 & ±0.16 respectively. After comparing the MDA level among case (IDDM & NIDDM patients) and control group, it is found that MDA level is higher in case group in respect to control group. Further, it is found that MDA level is more elevated in NIDDM patients in respect to IDDM patients.

DISCUSSION

This study was a cross-sectional study selected randomly & done in the department of Clinical pathology, PMCH, Patna. In both groups of DM, S. MDA levels were significantly higher than the normal according to statistical calculations mentioned in above tables DM comprises of a group of disorders that share a phenotype of hyperglycaemia. The complications are an important cause of morbidity & mortality in Diabetic patients. These complications are results of interaction of multiple metabolic, genetic & other factors.

SUMMARY AND CONCLUSION

The possible reason for significantly high S.MDA level in case of diabetes is due to increased blood glucose resulting in increased rate of production of free radicals.

CONCLUSION

MDA is increased significantly in both types of DM but more in case of NIDDM.

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